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Bronchoscopy.—Originally, the bronchoscope was devised for the removal of foreign bodies; to-day, bronchoscopy is an important supplemental method in the diagnosis and treatment of bronchopulmonary diseases. Its chief indications are hemoptysis, thoracic tumors, lung abscess, asthma, tuberculosis, bronchial obstruction and bronchiectasis. In certain conditions (heart disease, aortic aneurysm, pulmonary embolism, emphysema, bronchopneumonia, inflammatory lesions of the larynx, etc.) bronchoscopy is contraindicated or else not sufficiently informative. In 106 cases of hemoptysis in which X-ray examination was negative, bronchoscopy revealed the cause of the bleeding; in 34 additional cases bronchoscopy, too, was negative. The diagnosis of thoracic neoplasms has been greatly furthered by the use of bronchoscopy. Unfortunately, the suspicion of malignancy frequently arises too late for surgical treatment; hemoptysis is usually a late symptom. In 75 per cent of all tumor cases bronchoscopy leads to the correct diagnosis and it makes the differentiation and removal of benign tumors possible. In lung abscess, drainage can be promoted and the extent of the abscess determined by bronchoscopy. Many cases of lung abscess could be prevented if patients who vomited during an operation were immediately bronchoscoped. In some instances, bronchial asthma refractory to medication may be benefited by the removal of tenacious secretions. Preoperative bronchoscopy is indicated in pulmonary tuberculosis in order to exclude tracheobronchial lesions. Following surgical treatment, the bronchoscopist may be able to find the cause of persistent positive sputum. Bronchial obstruction of whatever cause calls for bronchoscopy, both as a diagnostic and as a therapeutic procedure.—*The Diagnostic and Therapeutic Possibilities of Bronchoscopy, G. J. Taquino, New Orleans M. & S. J., January, 1945,* **97:** *291.—(P. Lowy)*

Treatment of Tuberculous Bronchitis.—Local treatment with a 10 to 15 per cent solution of silver nitrate is advocated for nonstenotic tuberculous bronchial lesions occurring in the course of pulmonary tuberculosis. For disease refractory to bronchoscopic treatments or in stenotic bronchial lesions, collapse therapy is advised, thoracoplasty if the condition of the patient permits, pneumothorax being the second choice. Phrenic nerve operations are contraindicated. When the bronchus to the middle or lower lobe is involved, lobectomy may be considered in place of thoracoplasty. Occasionally cavitary drainage may be attempted.—*El tratamiento de la tuberculosis pulmonar con bronquitis tuberculosa asociada, R. Vaccarezza & A. Bence, An. Cáted. de pat. y clin. tuberc., December, 1943,* **5:** *263.—(R. Kegel)*

Tuberculous Empyema.—Among 4,527 patients with pulmonary tuberculosis treated in the Buenos Aires tuberculosis services between 1938 and 1943 there were 53 cases of tuberculous empyema, an incidence of 1.1 per cent. Of these 53 cases, 11 occurred spontaneously during the course of pulmonary tuberculosis, 42 arose during a therapeutic pneumothorax and 6 followed pneumonolysis. The incidence of empyema in 1,332

patients with pneumothorax was 3.1 per cent and in 513 with pneumonolysis 1.1 per cent. Broncopleural fistula was found in 68 per cent of the patients with empyema. Three clinical forms of empyema are recognizable, the simple, the toxemic and the malign. The last two are subdivided into pure and mixed infections. The kind of treatment depends upon several factors: the empyema itself, the status of contralateral or homolateral pulmonary lesions and the general condition of the patient. Medical treatment, aspirations and lavage, is tried first with thoracotomy and thoracoplasty in reserve. Of 39 patients treated, favorable results were obtained in 11 (28 per cent). There were 5 cures, 3 by thoracoplasty and one each by aspiration and by lavage. Seven patients were unimproved and 21 (54 per cent) died.—*Empiema pleural tuberculoso, J. Peroncini, R. Cucchiani & J. Niemetz, An. Cáted. de pat. y clin. tuberc., December, 1943, 5: 298.*—(*R. Kegel*)

Surgery of Tuberculous Empyema.—Since 9 of 10 patients with tuberculous empyema ultimately require one or another form of tuberculous intervention, the surgeon should be consulted at the onset of the disease so that the most favorable time for surgical treatment may not be missed. Early operations obviate the concomitants of late empyema, namely pachypleuritis and amyloidosis. In simple empyemata medical treatment, including aspiration and lavage, rarely cures empyemata. For aspirations a curved-aspirator inserted under fluoroscopic control is advocated. The patient should not be considered cured while an internal fistula is present. Most empyemata require an initial thoracotomy followed by a plastic operation. —*Consideraciones quirúrgicas sobre el empiema pleural tuberculoso, O. Vaccarezza, An Cáted. de pat. y clin. tuberc., December, 1943, 5: 310.*—(*R. Kegel*)

Tuberculous Meningitis.—Authentic cases of recovery from tuberculous meningitis are rare. A 16 year old boy became ill with typical clinical meningitis. Tubercle bacilli were demonstrated in his spinal fluid on smear and on culture. He made a complete recovery and was well seventeen months after the illness. The isolated bacilli were tested further and proved to be human tubercle bacilli of low virulence by animal inoculation. The second patient was a 41 year old woman in whom the diagnosis of tuberculous meningitis was made on the grounds of clinical meningitis and the finding of tubercle bacilli on smear and culture. Unfortunately the organisms were not tested as to their type and virulence and the patient was lost sight of.—*Recovery from Tuberculous Meningitis, G. H. Jennings, Lancet, April 14, 1945, 248: 466.*—(*H. Marcus*)

Meningoencephalitis in Hutinel's Disease. —A case of Hutinel's disease (pericardial and perihepatic symphysis) is reported. The interesting features in this case were the absence of free ascites and of liver cirrhosis and the rather rare complication of a meningoencephalitis in the right fronto-parietal lobe, accounted for clinically by a left-sided hemiplegia and paralysis of the facial nerve of the central type. The absence of liver cirrhosis can be explained by the cerebral complication occurring in the course of a generalized hematogenous dissemination, that terminated the life of the patient before a cirrhosis could develop. The nature of the cerebral localization proved to be tuberculous by the finding of numerous tubercle bacilli. Histologically there was only an acute nonspecific inflammatory process.—*Las complicaciones hemiplejicas meningoencephalicas de la enfermedad de Hutinel, P. H. Cantonnet, H. Lieutier, C. Perdomo, R. Radice, H. Castiglione & J. Medoc, Rev. de tuberc. d. Uruguay, 1944, 12: 73.*—(*L. Molnar*)

Tuberculin Sensitivity and Desensitization in Renal Tuberculosis.—The threshold of sensitivity to tuberculin was studied in a series of cases of genitourinary tuberculosis. The most reliable and safest method was found to be the one proposed by Liebermeister: sensitivity to tuberculin was tested start-

ing from the smallest dose of 0.1 cc. of a solution 1:1,000 billions, expressed as $10-^{16}$, and using for successive tests doses ten times larger ($10-^{15}$, $10-^{14}$, $10-^{13}$, etc.). The sensitivity to tuberculin was determined in 50 cases of unilateral kidney tuberculosis before surgery, or bilateral disease, in which surgery was contraindicated; in 15 cases of kidney tuberculosis after nephrectomy and finally in 10 cases of different urological involvement. There was a marked hypersensitivity in all cases of kidney tuberculosis. Patients with a low sensitivity (for instance $10-^{3}$) have, in general, a better prognosis than those who are hypersensitive (for instance $10-^{15}$). It is usually not difficult to differentiate a case of hypoergy, indicating a favorable immunological condition, from another where the low degree of sensitivity is an indication of terminal failure of the immunological situation. Generalization of tuberculosis, following nephrectomy, may be due to bacillemia caused by manipulation of tuberculous tissues, or to the reactivation of latent foci as a result of a failure in the defense mechanisms provoked by the surgical trauma. The desensitization to tuberculin prior to surgery or following surgery could, theoretically, influence the incidence of postoperative generalization. The first step towards desensitization is the determination of the threshold of sensitivity to tuberculin. Then 0.1 cc. of the threshold dose is injected intracutaneously twice a week, using increasing doses (tenfold), after each dose has been tolerated without any local reaction. A similar but somewhat slower course of desensitization could be followed in inoperable (bilateral) kidney tuberculosis. The relatively small material and the short period of observation do not warrant definite conclusions as to the effectiveness of this procedure. Desensitization after nephrectomy was employed as an attempt to control a residual cystitis; the results were, however, very uneven. Desensitization may sometimes be impossible, as in cases of so-called "fixed allergy."—*Sensibilidad y desensibilización tuberculinicas en la tuberculosis renal, L. C. Delatte & M. M. Diez, Rev. españ. de tuberc., October, 1944, 8: 744.*—(*L. Molnar*)

Genital Tuberculosis.—Genital tuberculosis is always a diffuse canalicular tuberculosis and a specific epididymitis is therefore nothing but the most easily detectable localization of a tuberculous process, secondary to a hematogenous, lymphatic or canalicular spread from the lungs or from the kidneys. That is why deeper lesions often persist after epididymectomy. They are not rare, but very often ignored. Biopsy of the testicle in cases of tuberculosis of the epididymis reveals almost constantly characteristic tuberculous lesions. Epididymectomy is not a radical operation but a palliative one and should not be performed until the process has become stabilized. It is only a precaution to prevent further extension and aggravation of the tuberculosis of the testicle. Epididymectomy should not be performed when the genital tuberculosis is a secondary phenomenon of a severe visceral tuberculosis, when the diagnosis is uncertain or when the disease is in its acute stage. The condition of the kidneys must always be investigated before epididymectomy is performed, because often tuberculosis of the kidney coexists and if nephrectomy is feasible, the genital tuberculosis often subsides after eradication of the renal focus.—*Remarques sur la tuberculose génitale de l'homme, B. Fey & R. Couvelaire, Presse méd., August, 1944, No. 15, 226.*—(*G. Simmons*)

Tuberculosis of Rectum.—Tuberculous lesions of the anorectal region are always a consequence of tuberculosis of other organs, generally the lungs. Infection takes place by swallowing of sputum containing bacilli. Occasionally the infection may be caused via the lymphatics or the blood. All anorectal fistulae and abscesses observed by the author have been of tuberculous origin; 19.69 per cent of all patients with pulmonary tuberculosis had some kind of rectal disease. Among 1,300 tuberculous patients, 75 (5.76 per cent) had fistulae, 43 (3.3 per cent) had

abscesses. The other 10 per cent had some form of nontuberculous disorder. Fistulae and abscesses are more frequently found between the ages of 18 and 42 years. The prognosis depends upon the development of the primary infection.—*Tuberculosis pulmonar y proctologia, R. Borlenghi, Rev. Asoc. méd. argent., February, 1945, 59: 91.*—(*W. Swienty*)

Vesico-intestinal Fistula.—Case history of a 21 year old male, who six months after the onset of a bilateral pleurisy with effusion developed abdominal pain, ascites and fever. The temperature decreased, but after three months rose again, the patient lost weight, had frequent diarrheas and nightsweats. The abdominal pain was recurrent and followed by a desire to urinate. Urination was painful and frequently interrupted by the emission of bubbles of gas. The urine had a fecal odor and tests for urobilin and indican were positive. Only traces of albumen were present. Fecal material was found in the urinary sediment. The patient died two and one half months later. (No autopsy.) It is assumed, however, that this patient had a specific intestinal ulcer which had ruptured into the bladder.—*Ein Fall von Darmtuberkulose mit einer Blasen-Darmfistel, S. A. Duursma, Nederl. tijdschr. v. geneesk., 1942, p. 140.*—(*G. Simmons*)

Tuberculosis of Myocardium.—This is a rare localization of tuberculosis. Generally it is secondary to tuberculosis of the pericardium or of the mediastinal lymph nodes. It may be caused by direct invasion by the blood-stream or by the lymphatics. Most often it is encountered in miliary tuberculosis. It then has no great prognostic importance because the myocardial process is only one manifestation of the wide-spread general infection. In the caseous form great parts of the heart muscles may be destroyed, but still there may not be any special symptoms which would allow making the correct diagnosis *in vivo*. The prognosis depends upon the development of the primary lesion and on the site of the lesion in the myocardium. The case of a 17 year old Negro boy, who had been suffering for three years of swellings of various joints, is discussed. Shortly before his admission he suffered from dyspnea, pain, palpitation and cough. The only cardiac sign was a soft blowing murmur in the fifth intercostal space to the left of the sternum. The X-ray film showed enlargement of the heart and congestion of the lungs. Blood cultures and agglutination tests were negative. The sedimentation rate was 43 mm. after one hour. The patient improved slightly on salicylates but died one month later with the symptoms of meningitis. The findings of the cerebrospinal fluid were not typical for tuberculous meningitis. The tentative diagnosis was purulent meningitis and mitral stenosis of rheumatic etiology. On autopsy, caseous tuberculosis of almost all the heart muscle was found. The patient had tuberculous meningitis and generalized miliary tuberculosis. The localization in the heart muscle was secondary to the involvement of the mediastinal lymph nodes, whereas the general hematogenous spread originated later from the cardiac lesion.—*Tuberculosis del miocardio, F. Saffie S. & R. Valenzuela Garcia, Rev. méd. de Chile, March, 1945, 73: 233.*—(*W. Swienty*)

Tuberculosis of Myocardium with Embolism.—A 65 year old white woman entered the hospital because of sudden nausea, vomiting, sweating, salivation, weakness. The heart was found enlarged, the electrocardiogram showed partial A-V block and intraventricular block. Signs and symptoms of occlusion of the right femoral artery and later of the left femoral artery appeared. The patient died. The autopsy showed marked chronic fibroplastic myocarditis; mural thrombosis of the endocardium of the left and right ventricles of the heart; cloudy swelling of the myocardium; embolism of the aorta, the right and left common iliac arteries, the right and left external and internal iliac arteries, and the right and the left femoral arteries; recent infarcts of the kidneys, spleen

and left lung; marked fibrous stenosis of the anterior descending branch of the left coronary artery; chronic fibrous tuberculosis of the lungs, calcification of the tracheobronchial lymph nodes and of the parabronchial lymph nodes and gangrene of both legs and feet. Microscopic examination of the lateral wall of the left ventricle showed masses of granulation tissue composed of epithelioid cells, lymphocytes and giant cells. Some of the giant cells contained refractile radial inclusions. These radial inclusions probably are a crystalline form of fat. Two structures closely simulating acid-fast bacilli were found in these lesions. Tuberculous myocarditis with endocardial thrombosis is not infrequent, but embolism from these thrombi is rare.—*Embolic Thrombosis of the Abdominal Aorta with Tuberculous (Histologic) Lesions of the Heart Containing Giant Cells with Radial Inclusions, R. A. Beebe & G. H. Coleman, Am. Heart J., April, 1945, 29: 539.*—(*G. C. Leiner*)

Tuberculosis of Eye.—Certain cases of keratitis and iritis, accompanied by cervical lymphadenitis, may be the expression of an allergic manifestation, inasfar as toxins from these tuberculous lymph nodes may maintain or reactivate intraocular tuberculous foci. Irradiation or surgical removal of these lymph nodes may result in spectacular cures. Five cases of keratitis are reported in which cervical radiotherapy led either to cure or to considerable improvement of the eye condition. Similarly, arthritic processes may be maintained by toxins or waste products from tuberculous lymph nodes. Thus, one patient, suffering from iritis and rheumatoid arthritis of the wrist and fingers and presenting enlarged cervical lymph nodes, was remarkably improved after institution of cervical radiotherapy.—*Le traitement des tuberculoses oculaires et rheumatismales par suppression de foyers ganglionnaires, J. Brun, Presse méd., April, 1943, No. 14, 180.*—(*G. Simmons*)

Tuberculosis of the Trochanter.—Tuberculosis of the greater trochantic bursa is rare and usually occurs secondary to osteomyelitis of the trochanter. Not until 1904 was concomitant involvement of the trochanter recognized. Later Cone and Swindt stressed the importance of trauma as an etiological factor. In subsequent series reported, the majority of cases showed evidence of healed or active tuberculosis elsewhere in the body. Complete removal of all infected tissue and immobilization of the affected part is the treatment of choice. Clinically, tuberculosis of the greater trochanter and its bursa is unusual in children but occurs at any age and in either sex. Usually there is mild pain in the involved leg over a long period of time with intervals of quiescence. A history of trauma may precede the onset of symptoms. There is slight swelling over the trochantic area, tenderness on pressure, but, as a rule, no pain on weight-bearing. Hip motion is free. Often a draining sinus is present over the trochantic area. Recurrences are common, some showing extension of the disease. The first X-ray evidence may be a small fleck of calcium in the bursa or a minimal area of destruction in the outermost part of the trochanter. The usual X-ray bone technique may not reveal the lesion. Therefore, Donovan and Sosman recommend the soft-tissue technique used to demonstrate calcium around the shoulder. The usual roentgenological finding in long standing cases is an area of destruction in the great trochanter with osteoporosis of the adjacent bone and some soft tissue swelling with calcium deposits in the bursa. Tuberculous involvement of the bursa must be differentiated from simple inflammatory bursitis which presents more acute symptoms and responds to simple treatment. Involvement of the trochanter requires differentiation from tumors and nonspecific osteomyelitis. Four cases of tuberculosis of the trochanter and its bursa are presented.—*Tuberculosis of the Greater Trochanter and Its Bursa, P. C. Briede, Radiology, January, 1945, 44: 32.*—(*G. F. Mitchell*)

Ultraviolet Radiation Control of Airborne Infection.—In the sleeping quarters of naval

recruits arranged in two groups of buildings of 11 each on both sides of a central training field, ultraviolet lamps of high and low intensity were installed in half of the buildings. The other half served as controls. Within these quarters a combination of upper air and floor irradiation was employed. The level of ultraviolet intensity was a determining factor in the control of airborne infection. A reduction of 25 per cent in respiratory illness resulted in those barracks equipped with high intensity radiation. There was no appreciable reduction in the rate of illness in the barracks equipped with low intensity irradiation. This reduction was most noticeable in the early winter months when the rate of illness was high throughout the camp. Bacterial counts in the irradiated quarters showed a 50 per cent reduction in the bacterial population of the air as compared with that in the nonirradiated control barracks.—*Ultraviolet Control of Air-borne Infections in a Naval Training Center, S. M. Wheeler, Lt. (MC) USNR, H. S. Ingraham, Lt. (MC) USNR, J. Gershon-Cohen, Lt. Comdr. (MC) USNR, & E. W. Brown, Capt. (MC) USN, Am. J. Pub. Health, May, 1945, 35: 457.*—(*M. B. Lurie*)

Pathology of Atypical Pneumonia.—The morbid anatomical changes in 42 cases from the U. S. Army of acute interstitial pneumonia (the so-called atypical pneumonia, etiology undetermined) are described. Of these, 21 were uncomplicated by superimposed secondary bacterial infection. The bronchioles revealed an acute focal bronchiolitis with early ulceration and, in some instances, complete desquamation of the mucous membrane. The bronchiolar lumina contained frank pus mixed with desquamated and disintegrated cells; no bacteria could be found in most cases. The walls of the bronchioles were dilated, sometimes markedly, even in the cases in which death occurred early, but complications such as chronic bronchiectasis were not seen and necrosis of the walls was seen in only one instance. The walls were infiltrated chiefly with mononuclear cells extending radially into the regional interstitial tissues of the lung, and in some cases there was fragmentation of the smooth muscle, elastic and reticulum fibres. The larger and medium sized bronchi showed only submucosal edema and congestion with a scattering of mononuclear and polymorphonuclear cells; focal ulceration was noted in only two instances. The alveolar walls were most severely involved close to the affected bronchioles; the lumina frequently contained air although focal areas of atelectasis were seen. In some instances there was extensive desquamation of alveolar lining cells and less frequently serous exudate with or without hyaline membrane formation. Usually no polymorphonuclear leucocytes were seen in these areas, and no microörganisms were found. Hemorrhages, when present, were usually terminal and were limited to those alveoli close to involved bronchioles. In the presence of secondary bacterial infection such as frank bronchopneumonia, lobar pneumonia and pulmonary abscess, it was sometimes possible to distinguish areas of acute interstitial pneumonitis in adjacent portions of the lung. The similarity of the lesions to those seen in measles pneumonitis and in epidemic influenza, especially when death occurs within the first five days of illness, together with the failure to demonstrate consistently any organisms in the lung tissue, suggests that a virus may be the etiological agent; it is pointed out, however, that acute interstitial pneumonitis is also seen in pertussis, a bacterial infection. In the cases in this series the leucocyte count tended to be high with 70 to 98 per cent polymorphonuclear cells; all of these cases came to autopsy, however. Cyanosis was common, and was best explained by the massive plugging of the smaller bronchial branches, the interstitial infiltration of alveolar walls and the pulmonary congestion. The presence of hyaline membranes was too inconstant to be the sole explanation.—*Pathologic Anatomy of "Atypical Pneumonia, Etiology Undetermined": Acute Interstitial Pneumonitis, A. Golden, Arch. Path., October, 1944, 38: 187.*—(*D. G. Freiman*)

Etiology of Atypical Pneumonia.—The present status of the etiology of primary atypical pneumonia is reviewed. A variety of known agents, including bacteria, fungi, Rickettsia and viruses, can produce this clinical syndrome, but accounts for a very small proportion of the cases diagnosed. The cause of the remainder remains to be identified. The results of extensive animal experimentation have been difficult to interpret owing to the lack of a truly susceptible animal and the occurrence of spontaneous diseases in the animals employed; there has as yet been no confirmed report of the isolation in animals of an agent definitely related immunologically to the human disease. The variety of immunological reactions demonstrable in convalescent sera, including cold hemagglutination, fixation of complement with various dissimilar antigens but especially fresh tissue suspensions, the prevention of the development of antibodies to the mouse pneumonia virus, and the agglutination of an indifferent streptococcus can be attributed to a nonspecific alteration in the serum proteins and their reactivity as well as by common antigens. Because of these equivocal findings, experiments were conducted with human volunteers. In the first experiment, sputa and throat washings from cases of atypical pneumonia were sprayed into the noses and throats of 12 men; of these, 2 failed to become ill, 2 developed mild infections without fever and 3 developed moderately severe atypical pneumonia. Between these extremes there were cases resembling the disease but without demonstrable pulmonary infiltration; these cases were similar to those diagnosed as "bronchitis resembling atypical pneumonia" or "suspected atypical pneumonia." In the second experiment 36 men were isolated, great precautions being taken to avoid cross infection and extraneous exposure. They were divided into three groups, the first being inoculated with 10 cc. each of pooled sputa and throat washings from 7 cases of atypical pneumonia, the second with a similar amount of this same material bacteriologically filtered, and the third with this same material autoclaved. All of the first group, 9 of the second and 4 of the third became ill either with primary atypical pneumonia or minor respiratory illness of undifferentiated type; however the cases of pneumonia were about equally distributed in all groups (3 in the first, 4 in the second and 3 in the third). No conclusion regarding infectivity of the filtrates could therefore be drawn and the question of nonspecific evocation of a latent agent or inadvertent infection was raised. The experiment was therefore repeated redoubling precautions against inadvertent infection and increasing the size of the control group receiving autoclaved sputum to 18. The inoculum now consisted of pooled sputa and washings obtained from 6 of the cases from the second experiment, thus providing an opportunity for passage of the agent On this occasion the results in groups one and two were the same, with 3 cases each of atypical pneumonia and 5 cases each of undifferentiated respiratory illness; 4 cases in each group failed to become ill. In the control group there was only one case of mild respiratory illness in a man known to have broken isolation; all the rest remained well. These results suggest that primary atypical pneumonia is initiated, if not caused by a filtrable agent, presumably a virus, and that a latent agent is not evoked nonspecifically. A difference in incubation periods, almost twice as long in those cases receiving filtered inoculum, suggests the possibility that bacteria may act in conjunction with the filtrable agent; the possibility that filtration causes some loss of the infecting agent cannot be ruled out however.—*The Present Status of the Etiology of Primary Atypical Pneumonia: Commission on Acute Respiratory Diseases, J. H. Dingle, Director, Bull. New York Acad. Med., May, 1945, 21: 235.*—*(D.G. Freiman)*

Atypical Pneumonia.—One hundred and forty-four cases of primary atypical pneumonia in ambulatory patients were studied. Pleural involvement was found in 21 patients (14.5 per cent), in 11 of whom no parenchymal lesion could be demonstrated. In only 3

cases was the site of the pleurisy the costophrenic sinus, while in the rest the pleuritic process involved the lower portion of one of the long fissures. The anatomy and roentgenology of the interlobar fissures are described and illustrated in detail. Clinically, the majority of the 144 patients had only mild, non-incapacitating symptoms (cough, slight expectoration, malaise, chilliness) and there was no difference, in this respect, between the patients with and without pleural involvement. Physical signs were scant and consisted of diminished or exaggerated breath sounds, slight dulness, subcrepitant and/or rhonchial râles and occasional pleural friction rub. The temperature, pulse, urine, leucocyte count and differential count were normal in most cases. Roentgenographic signs of pneumonia or pleurisy usually disappeared in two to four weeks. Involvement of the lower end of one of the long fissures presented itself in the frontal view as a small area of homogeneous density in the lower lung field extending down to the cardiophrenic angle and not reaching beyond the midclavicular line laterally. In the absence of pneumonia the lung markings were normal. In the lateral view, a band-like or fusiform density could be seen near the junction of the diaphragm and the anterior chest wall. In the oblique (right anterior for the left and left anterior for the right long fissure) films a homogeneous triangular shadow in the anterior portion of the lung field indicated involvement of the lower end of the interlobar fissure. In the differential diagnosis, pneumonia or atelectasis of the right middle lobe, pneumonia in the lower anteromedial portion of the left upper lobe, and infiltrations in the lower lobes must be excluded. Middle lobe pneumonia presents a triangular, and not a band-like, appearance in the lateral view. Pneumonic processes located in the lower medial portion of the left upper lobe do not extend down to the diaphragm in the postero-anterior film, while in the lateral view they show up as a broad area of density. Lower lobe infiltrations can be distinguished from interlobar pleurisy by the lesser homogeneity and posterior location of the former. The high incidence of pleural involvement in the authors' series of atypical pneumonias is partly attributed to the frequent use of lateral and oblique views.—*Pleuritic Involvement Associated with Primary Atypical Pneumonia: A Roentgenographic and Clinical Study, A. L. Bachman, N. O. Sara & H. E. Mantz, Am. J. Roentgenol., March, 1945, 53: 244.*—(*P. Lowy*)

Friedlander Bacillus Pneumonia. — The authors have compiled 287 cases of pneumonia caused by Friedlander bacillus from the entire world literature from 1882 to 1941; 233 of those with a mortality rate of 94 per cent were acute and 54 with a mortality of 30 per cent were chronic forms. (The cases of Zander (411) and Bathmagar and Singh (13) were disregarded as without exact control.) It is difficult to distinguish between the *Klebsiella*, *Echerichia* and *Aerobacter* groups. There even exist intermediatory forms between these different groups. The antigen contained in the capsule of *Klebsiella pneumoniae* type B, is similar to the capsule antigen of pneumococcus type 2. Although rare, some forms of *Klebsiella* can pass from the gram-negative to a gram-positive stage. Friedlander bacillus and all of the *Klebsiella* are habitual inhabitants of the respiratory system of man and it has been found that the localization in the tonsils is the most frequent. The authors have treated 244 cases of pneumonia of which 29 (11.8 per cent) were caused by *Klebsiella*. In 15 cases the pathogenic agent was *Klebsiella pneumoniae*. In 8 it was *Klebsiella ozaenae* and in 6, *Klebsiella capsulata*. All the germs were found in the sputum and the diagnosis was made only after at least six positive cultures were obtained. In 10 cases pneumococcus was associated with the *Klebsiella*, in 4 others, tubercle bacilli. Never was fungus found in association with the *Klebsiella*. The infection was always bronchogenic and generally started from the tonsils. Predisposing factors were colds, alcoholism, malnutrition, physical exhaustion and infection of the respiratory tract. Sixteen acute pneumonias and one

acute bronchopneumonia were studied. The acute form generally started abruptly. Herpes was rare. The patients were cyanotic and dyspneic. The age of the patients was from 18 to 64, with an average of 37.7 years. The mortality was 4 (25 per cent). Death occurred in ten to twenty-eight days. The average age of these patients was 48.8 years. Fifteen cases presented localization in the inferior lobes. Only one involved the upper left lobe. Fever and sputum were atypical. In 2 cases septicemia, purulent meningitis and pleuritis were found. Three cases showed leucopenia with shift to the left and monocytosis. Of 2 cases with lymphopenia, one died. Five had leucocytosis and shift to the left. Of those, 4 had monocytosis and one monocytopenia. Tubercle bacilli were not found in any one of the acute cases. The treatment consisted of sulfathiazole. In one case it was combined with penicillin. The duration of the disease was from five to thirty-seven days with an average of 17.7 days until full recovery. Twelve chronic forms were also studied. In 4 cases pneumococci and in 4 Koch bacilli were found associated with the Friedlander bacillus. The age was from 27 to 71 years with an average of 46.1. One 31 year old patient died sixteen months after onset of the disease. The start of the disease was abrupt in 7 and insidious in 5. The clinical picture is easily mistaken for tuberculosis. In 4 cases pulmonary abscesses were present. In 2 others tuberculous cavities appeared during the disease. Eight patients had leucocytosis with shift to the left; 5 of them had monocytosis. Four other patents had a normal white count. One patient recovered after forty-three days. All the others remained unchanged from three months to ten years. Death is generally due to toxemia. There is an intense reaction of the pulmonary tissue to this toxemia which is followed by necrosis and persistent pulmonary edema. Necrosis and hemorrhages are common in the acute forms. Bacteremia is almost always present in the acute forms of Friedlander pneumonia, but it is generally not followed by spread to other organs. For example, meningeal complications are rarely seen. The bacteremia has no prognostic significance. Relative or absolute leucopenia generally gives a poor prognosis. In the X-ray picture four different phases of the development of the disease can be seen: First, beginning bronchopneumonia; second, secondary pseudolobar coalescence; third, formation of multiple abscesses and thin walled cavities; fourth, fibrosis. In differential diagnosis of the acute forms pneumococcus pneumonia can be ruled out by the absence of herpes, the tendency to bilateral involvement, the abscess formation and the bacteriological examination of the sputum. Pulmonary gangrene also has to be excluded. Complications are pulmonary abscess, empyema, pleuritis, septicemia and meningitis. The chronic form has a relatively benign course over months or years. It often simulates pulmonary tuberculosis. The X-ray picture is always suspicious of tuberculosis, and association with tuberculosis is not infrequent. The cavities are thin-walled, and contain a purulent hemorrhagic fluid. The differential diagnosis has to exclude tuberculosis, chronic bronchitis, bronchiectasis and multiple lung abscesses. Complications are pulmonary abscess, empyema, bronchiectasis, hemoptysis, phlebitis, arthritis and endocarditis. The treatment in the acute and chronic forms consists of the sulfanilamides, good nutrition and rest. Penicillin seems to have no effect. The high percentage of recovery, especially of the acute forms, is due to the modern treatment.—*Neumopatias a Friedlander, F. Hermosilla D., S. Marin Tagle, L. Parades & E. Bellolio Z., Rev. méd. de Chile, January, 1945, 73: 36.*—(*W. Swienty*)

Treatment of Empyema.—More than 90 per cent of all the pneumococcal pneumonias are pleuro-pneumonias. Empyema generally appears when the disease is in the phase of grey hepatization. A portion of the parenchyma may soften and an abscess form. The evacuation of the pus may take place by vomica. But often the pus is evacuated into the pleural cavity, especially if not too

many adhesions are present. Generally, postpneumonic empyema is observed in patients who had no adequate medical care or who present a definite lowering of resistance towards the invading agent. In some cases, the empyema heals spontaneously by absorption or even by vomica. The empyema does not retard the healing of the parenchymatous lesions. Occasionally, it may extend and provoke mediastinal, subphrenic or pericardial abscesses. Therapeutically, it is of the utmost importance to avoid all maneuvers that could interfere with the respiratory equilibrium of the hemithorax. Surgical methods that bring the pleural cavity in contact with the air produce irritation and delay cicatrization by apposition of the two pleural folds. The resistance of the pleura to the infection becomes diminished by contact with air. The authors describe a medical treatment of empyema. The aspiration of pus is followed by a lavage of the pleura with luke-warm physiological saline solution in large amounts (one to three quarts). This is repeated at intervals of forty-eight hours. After X-ray studies show reduction of the empyema the lavages are done at longer intervals. Small transfusions of whole blood from 100 to 150 cc. are given to maintain the resistance of the patient. The authors have treated a number of cases this way. In their latest report they have added 20,000 to 40,000 units of penicillin to the saline solution; 5,000 to 15,000 units of penicillin were given intermuscularly every four hours for one to two days. In the 2 cases so treated complete cure was obtained in forty-five days. In a third case 30,000 to 35,000 units of penicillin were injected intrapleurally without pleural lavage every twenty-four hours and after four days every forty-eight hours. The pus became sterile, but the fever persisted. Seven days after the last intrapleural administration of penicillin the pus was evacuated by pleural puncture and after lavage with saline 80,000 units of penicillin were instilled intrapleurally. Complete cure was obtained in twenty-six days. In all 3 cases the pus was found to be sterile twenty-four hours after application of the penicillin. The closed lavage of the pleural cavity helps to eliminate the toxic products of the suppuration and prevents mixed infection so often occurring in pyopneumothorax. Penicillin should be used intrapleurally; 50,000 units should be injected intrapleurally every twenty-four hours for four days and every forty-eight hours thereafter until afrebility is obtained. In the presence of other inflammatory processes additional intramuscular administration of 20,000 units of penicillin every four hours may be necessary.—*Tratameinto medico del empiema pleural metaneumonico, R. Gondar, F. Saffie & J. Bonell, Rev. méd. de Chile, January, 1945, 73: 30.—(W. Swienty)*

Local Penicillin in Lung Abscess.—A chronic putrid lung abscess had been treated with intramuscular penicillin without marked benefit. The patient continued to go downhill and hemorrhages continued. The abscess was located in the apical portion of the right lower lobe. The organisms involved were predominantly *Streptococcus viridans.* Also present were *N. catarrhalis,* fusiform bacilli, pneumococci and two other strains of streptococci. Because of the desperate condition of the patient, local administration of penicillin was decided upon. Thick pus was aspirated from the chest at a point about 2 inches from the midline posteriorly, in the seventh posterior intercostal space. At a depth of 8 cm. thick pus was encountered and 20,000 units of penicillin in 5 cc. of saline was instilled. On following days different spots were chosen for the injection after preliminary aspiration of pus each time. The condition improved remarkably. After the first injection the temperature became normal. However, although the symptoms were controlled by this treatment the abscess as seen on X-ray film did not close. Surgery was decided upon, and after resection of two ribs the abscess cavity was located and a drain placed. The interlobar fissure was tremendously thickened and contained pus. The patient died shortly after the operation.

Autopsy shows acute peritonitis resulting from an apparently rather silent gastric ulcer. It is felt that this patient was benefited by the local application of penicillin, and benefit might have been more apparent had he been treated in this fashion more promptly.—*Local Instillation of Penicillin in Lung Abscess, D. Pickering & R. Greenville-Mathers, Lancet, April 28, 1945, 248: 530.*—(*H. Marcus*)

Surgical Treatment of Lung Abscess.—Eighty-five cases of lung abscess were treated surgically with a mortality of 16.5 per cent. External drainage was performed in 77 cases: in 3 cases lobectomy was done (2 deaths); 3 apical thoracoplasties were performed (2 good and 1 doubtful result); in one case Monaldi's aspiration with subsequent apical thoracoplasty was done. In 3 cases the abscess drained spontaneously through the bronchus after institution of a pleural tamponade, preliminary to further surgery. In 15 of these cases the condition had persisted for more than one year and in this group there were 13 deaths. In the group of cases that were operated on soon after the onset of the condition, there were 2 deaths (one due to an air embolus and one due to a secondary pyopneumothorax). In 3 cases a bronchocutaneous fistula persisted after external drainage. No fistulae occurred in cases operated on less than three months after onset of the condition. No case of bronchiectasis was observed in lung abscesses of less than three months' standing. Early surgery, therefore, not only gives a smaller death rate but prevents occurrence of secondary complications. Recurrence of a lung abscess occurred in 2 cases, four and eighteen months after institution of external drainage.—*Les résultats du traitement chirurgicale de l'abscès du poumon: A propos de 85 observations, P. Sauty & M. Bérard, Presse méd., August, 1944, No. 15, 225.*—(*G. Simmons*)

Penicillin Treatment of Pulmonary Suppuration.—Local and systemic treatment with penicillin was tried in hemothorax, pyogenic empyema, mixed infection tuberculous empyema, extrapleural suppuration and pulmonary suppuration. Intrapleural instillation of penicillin into uninfected hemothorax cases apparently prevented infection. However, deposition of fibrin goes on regardless of treatment and, when this becomes extensive, rib resection and evacuation of fibrin and clot become necessary. Postoperatively penicillin appeared to be of value in preventing infection. Pyogenic empyema is almost never cured by aspiration of pus and instillation of penicillin. Although the pus may become sterile, a rigid pleural space results, and the lung does not reëxpand. Thoracotomy and drainage became necessary. Penicillin again is valuable following operation when instilled into the pleural cavity. Administration of penicillin into the pleural cavity may be of value before an empyema becomes thick, and if the underlying lung process is still active. This is especially true of hemolytic streptococcus infections. In secondarily infected tuberculous empyemata penicillin treatment has been of distinct value. Although drainage may eventually become necessary, all patients were benefited with return of temperature to normal and disappearance of the secondary invaders from the pleural exudate. The single dose is 20,000 to 50,000 units given intrapleurally at intervals from one to four days. The total dose is up to 400,000 units. Penicillin is useless if the only infecting organism is penicillin resistant, but if several organisms are present, one penicillin sensitive and one resistant, distinct improvement has resulted, with eventual disappearance of both organisms. Extrapleural infections following upon surgery of the thorax are successfully treated if the organism is sensitive. The cases of intraplulmonary suppuration fall into several groups. Infection with nonhemolytic microaerophilic streptococci is fairly common. The organism is penicillin sensitive, but, although the infection of the lung can be treated successfully with intramuscular administration of 60,000 to 120,000 units of penicillin for about two weeks, complications have been

frequent. Empyema could not be aborted, and cerebral abscesses have occurred in 3 out of 7 such cases. Pulmonary suppuration due to mixed anaerobic infection has not been treated successfully in spite of the fact that the organisms were penicillin sensitive. Surgical drainage became necessary in 5 out of 6 cases treated with penicillin. Two cases of actinomycosis were also treated. Good results were obtained in one case, but treatment was protracted and the dosage high. This patient received one course of 1,800,000 units over a course of six weeks. Three weeks later he had a recurrence and another course of penicillin was given. The second course lasted twenty-eight days, and 5,600,000 units were given. The patient remained well for a four-month period.—*Pleural and Pulmonary Suppuration Treated with Penicillin, J. E. H. Roberts, O. S. Tubbs & M. Bates, Lancet, January 13, 1945, 248: 39.*—(*H. Marcus*)

Absorption of Aerosol Penicillin.—The absorption of penicillin by the lungs was studied experimentally. Solutions of penicillin containing 10,000 units of penicillin per cc. were dispersed in a room of 3,000 cubic feet by means of an electric generator. Twenty cc. of a solution of sodium penicillin in water could thus be dispersed in an hour. An extremely fine mist was produced. Blood agar cultures which had previously been inoculated with 500 colonies per plate of staphylococcus aureus showed complete inhibition of growth if the plate was exposed in the room at a distance of 25 feet from the generator for fifteen minutes at the start of the experiment and for five minutes towards the close of the hour. Considerable quantities of penicillin were noted in the blood and in the urines of volunteers who were exposed to the aerosol penicillin. After thirty minutes' exposure in the room, one part of urine was sufficient to inhibit the growth of staphylococcus aureus when added to 19 parts of a broth culture. The possible practical applications of these experiments are obvious. It has been demonstrated that aqueous sodium penicillin remains active for a considerable time when dispersed as an aerosol. The dispersion of penicillin in entire hospital wards, operating or dressing rooms thus becomes a practical procedure. The administration of penicillin to patients who object to both intramuscular injections or to the use of face masks, or the administration to extremely young patients, can be satisfactorily solved in this manner.—*Absorption of Aerosol Penicillin via the Lungs, F. A. Knott & W. H. Clark, Lancet, April 14, 1945, 248: 468.*—(*H. Marcus*)

Penicillin by Inhalation.—Experiments were conducted in 5 normal subjects to determine the feasibility of administering penicillin via the respiratory tract by means of nebulization. A Collison nebulizer was used and oxygen passed through it at the rate of 8 to 10 liters per minute. The concentration of penicillin was 80,000 units per cc. The mist was delivered by means of the nose piece of the standard BLB mask. The patient inhaled through this and was instructed to exhale through the mouth. At least 40,000 units can be absorbed during a half-hour period, and effective blood concentrations can be obtained. Maximal excretion followed during the half hour after the conclusion of the experiment. The waste of material is high, amounting to from 60 to 75 per cent of the material. It is possible, however, that weaker solutions, such as 5,000 units per cc., might be employed to equally good advantage, thus making the procedure practicable for treatment of infections of the lower respiratory tract. Aqueous calcium penicillin was used because sodium penicillin solution was found too irritating.—*Penicillin by Inhalation, N. Mutch & R. E. Rewell, Lancet, May 26, 1945, 248: 650.*—(*H. Marcus*)

Bronchiectasis following Atypical Pneumonia.—Primary atypical pneumonia, etiology undetermined, is usually a mild to a moderately severe illness affecting young adults. The duration of the disease is from five to fourteen days; it is uninfluenced by

chemotherapy. The literature of the clinical, roentgenological and pathological findings of atypical pneumonia is reviewed. Two factors responsible for the development of bronchiectasis may be present in atypical pneumonia, bronchial infection and bronchial obstruction. Twenty patients with bronchiectasis following attacks of atypical pneumonia during the winter of 1942–1943 were seen. In all of them, roentgenograms taken at the time of induction into the Army had been normal. These patients had had no symptoms prior to the pneumonia. There was no spontaneous healing of the atypical pneumonia; productive cough, râles and roentgen signs of unresolved pneumonia persisted. Bronchographic examination after several months revealed bronchiectasis. Bronchography was repeated over a period of two to six months. In 3 patients the bronchiectasis disappeared. Ten of the 17 patients had lobectomies with complete relief of symptoms, without operative mortality. Pathological examination of the resected lobes confirmed the diagnosis. Seven representative case histories are given.—*Bronchiectasis ollowing Atypical Pneumonia, E. B. Kay, Arch. Int. Med., February, 1945,* ***75:*** *89.—(G. C. Leiner)*

Bronchiectasis.—The following theories regarding etiology are listed: (*1*) preëxistent recurring bronchial infection, (*2*) chronic lobar or lobular pneumonia, (*3*) pertussis with pneumonia, (*4*) acute or chronic lung abscess, (*5*) bronchial obstruction due to carcinoma, (*6*) foreign body, (*7*) inflammatory cicatrix caused by specific infection, as tuberculosis, or caused by various nonspecific infections, and (*8*), to a lesser extent, chronic empyema or lesions causing gradual bronchial compression with retention of secretions and subsequent infection. The authors do not believe that sinus disease is frequently concomitant. Pathologically bronchiectasis is characterized by a degeneration of the bronchial wall with infection, the inflammation and necrosis of the tissue extending beyond the bronchial walls and, in advanced cases, producing bronchopulmonary suppuration. The diagnosis is based early on the presence of slight cough with scanty or moderate amounts of nonfoul sputum and, occasionally, hemorrhage. At this period the patient's general condition is good. Physical signs may be absent or, if present, may consist of diminished resonance and râles. Later, however, cough becomes severe, sputum is abundant and foul, clubbing is frequent, hemorrhage is by no means unusual, and, when drainage is impaired, there is severe sepsis. The complications are: (*1*) hemorrhage, which is quite common (Hedblom is cited as authority for the statement that hemorrhage occurs probably more often in bronchiectasis than in tuberculosis); (*2*) bronchopneumonia (the patient is usually not quite so toxic as in primary pneumonia and recovery is often more rapid); (*3*) atelectasis, which is due to obstruction by inspissated secretion or by the cicatrix of chronic inflammation (when atelectasis is permanent, there is produced the "shrunken lobe" of Jones and Cournand); (*4*) empyema. The authors point out that diagnoses on plain films are not consistent, but may be suspected from prominent bronchial markings, from rounded areas of increased radiance, the so-called "honeycombs," from lobar or lobular atelectasis, and from areas of mottled densities. The means of choice for diagnosis of bronchiectasis are bronchography and bronchoscopy with suction to remove secretions in those cases where visualization is not good due to inspissated secretions or cicatricial stenosis. Tuberculosis, foreign body, bronchogenic carcinoma and lung abscess must be differentiated from bronchiectasis. The authors present the following pathological classification: (*1*) ulcerative, the type in which hemorrhage is frequent; (*2*) stenotic, which favors the development of atelectasis and the progression of infection; (*3*) fibrotic, in which abundant peribronchial fibrosis exerts a traction mechanism; and (*4*) the "dry hemorrhagic" type not characterized by either cough or sputum but rather by recurrent hemorrhage. This latter type may be on a congenital basis, and

these cases often give a negative history. Both physical examination and X-ray may be negative. However, the bronchogram clinches the diagnosis. Anatomically the cylindrical type is most common and is most often found at the bases. The saccular is less common and occurs more often in the upper lobes. Occasionally there is a fusiform type. All cases reported in this study were confirmed by bronchogram except where lipiodol instillation presented difficulty or where atelectasis was evident, or neoplasm was suspected. Sixty-two cases were presented. There was a considerable incidence of antecedent respiratory disease. Chronicity was an important factor. Râles were present in 93 per cent. There was a history of hemoptysis in one-third. Clubbing and foul sputum were relatively infrequent. Fifty per cent of the lesions were located in the left lower lobe, 31 per cent in the right lower lobe, 12 per cent in the right middle lobe, and 16 per cent in both lower lobes. None occurred alone in either upper lobe. The incidence of bronchiectasis in the Army does not exceed that in civilians. It was present before induction. Mobilization regulations stipulate bronchiectasis as disqualifying. The C.D.D. board is authorized to recommend discharge on a Certificate of Disability, after which, if the individual is in need of hospital care, he is eligible for admission to a Veterans' Hospital. A group of case reports is presented. Of 1,753 cases observed at the Station Hospital since September, 1940, and separated from the service for various causes, 32 cases, or 2 per cent, were discharged because of bronchiectasis.—*The Diagnosis of Bronchiectasis: Clinical and Roentgenological Observations, M. H. Joress & S. A. Robins, Dis. of Chest, November–December, 1944, 10: 489.*—(*K. R. Boucot*)

Atelectasis and Bronchiectasis.—The decreased endothoracic pressure consecutive to bronchial obstruction and atelectasis is considered by many as the chief mechanism in the pathogenesis of bronchiectasis. In face of this tendency, it is useful to reconsider the question of the relationship between atelectasis and bronchiectasis. Experimental studies, clinical and roentgenological evidences have proved that with decreased endoalveolar pressure there is a tendency in the obstructed bronchus also to collapse and that this tendency cannot be counteracted by decrease of endothoracic pressure. Bronchial obstruction causes the bronchus to dilate only when it is incomplete; in this case there is no atelectasis, but obstructive emphysema. Infection is the most important single factor in the genesis of bronchiectasis. The inflammatory process causes deep changes in the various structures of the bronchial wall. The secondary mechanical factors act on the weakened bronchus in establishing an irreversible bronchial dilatation. The bronchial occlusion facilitates the development and persistence of infection, and the incomplete occlusion with a valve-mechanism in the presence of infection also favors the dilatation of the bronchi in the blocked segment. A superimposed infection can also cause a latent congenital or acquired bronchiectasis to become clinically significant. The rapidity with which bronchiectasis develops depends upon the association of the different factors, among which pleuropulmonary fibrosis should also be mentioned. The rôle of infection is emphasized by the observation that not all cases of atelectasis are followed by bronchiectasis. The higher incidence of bronchiectasis in children can be explained by the narrower bronchi, by the less powerful expectoration and by the more delicate anatomical structures in children. The sequence of events in the case of bronchiectasis associated with tuberculosis brings further evidence in favor of the above described pathogenetic view. Experimental studies also support this opinion. The prevention and treatment of bronchiectasis, therefore, consists essentially in the treatment of all infections of the respiratory tract (chemotherapy) and in all measures that prevent retention of secretions and bronchial obstruction, such as symptomatic treatment, postural drainage, removal of foreign bodies, bronchoscopic aspiration, appropriate

pre- and postoperative care, etc.—*Atelectasia pulmonar y dilatación bronquial-relaciones reciprocas, R. F. Vaccarezza & J. M. Leston, An. Cáted. de pat. y clin. tuberc., 1943, 5: 5.*—(*L. Molnar*)

Loeffler's Syndrome.—Two cases of transient pulmonary infiltrate with moderate eosinophilia are presented. The rapid disappearance of the infiltrations (two weeks) with complete *restitutio ad integrum* and the blood picture (4 per cent eosinophils, 26 per cent lymphocytes) excluded the possibility of a specific pulmonary involvement. No allergenic factors, such as the presence of ascaris in the feces, could be demonstrated. One of the patients, however, had tuberculous peritonitis and the other had been previously exposed to tuberculosis. It is believed that in both cases the transient infiltrate was the expression of an allergy against "tubercle toxins."—*Contributo allo studio dei cosidetti "infiltrati polmonari fugaci," V. Gramazio, Ann. Ist. Carlo Forlanini, 1942, 6: 237.*—(*G. Simmons*)

Therapy of Bronchial Asthma.—An attack of bronchial asthma can be produced experimentally in a guinea pig by exposing the animal to the inhalation of a finely dispersed solution of histamine. Such a "histamine-asthma attack" may well serve for the study of the prophylactic or therapeutic value of different substances. Dioxyephedrine is particularly efficacious against histamine asthma and the effect of this substance can be enhanced further by addition of diphenylpiperidicopropane.—*Experimentelles zur Asthmatherapie, O. Schaumann, München. med. Wchnschr., 1942, 11: 742.*—(*G. Simmons*)

Pulmonary Acariasis.—During a two-year period the authors discovered that bronchial asthma was responsible for 48 per cent of respiratory cases and 21 per cent of all cases invalided out of the Army from their medical division. In general, the observations on these cases showed (*1*) the presence of eosinophilia, in some cases of a very high degree; (*2*) essentially negative past or family history of asthma or other allergic manifestations; (*3*) exclusion of alimentary parasitism by the usual pathological tests and (*4*) poor response to the usual medical treatment of asthma. Because of previous reports of success, a search was made for mites in the sputum—and in 11 of 21 cases whose sputum was examined by a special technique the mites were found. They were mites of either *tyroglyphus* or *tarsonemus*. The radiological findings in these cases were typically those of fine, diffuse mottling, giving a ground glass appearance. Reëxamination of the lungs after arsenical treatment showed a clear parenchyma. The treatment consisted in arsenicals of the pentavalent variety and resulted in remarkable success—with a marked reduction in the eosinophilia as well as distinct clinical improvement. The disability was acquired after the inhalation of mite-laden air. The majority of the patients had been exposed to dust emanating from rice, cereals, flour, sugar, etc. A few had worked in linen and leather goods depots. The frequent existence of tyroglyphid and tarsonemid mites in stores and stored products has been recognized. In conclusion the authors state that the possibility that pulmonary acariasis may be responsible for other respiratory disorders besides asthma should not be ignored and that the treatment of all such cases, whether asthmatic or otherwise, by the administration of pentavalent arsenicals is worthy of consideration.—*Pulmonary Acariasis: A Possible Cause of Asthma, E. Soysa & M.D.S. Jayawardena, Brit. M. J., January 6, 1945, 1: 1.*—(*D. H. Cohen*)

Parasitic Diseases of Lung.—In 2 people who had had long contact with animals (dogs and foxes) multiple round shadows of the size of a small nut and containing an irregularly limited denser nucleus were seen, localized near the pleura. Clinically there was acute onset of dyspnea and acute bronchitis. Similar pictures had been described previously and it was believed that they were due to pulmonary localization of the ova of *Taenia*

solium. The author believes, however, that his 2 cases, as well as some of those described as cysticercosis, may have been due to the pulmonary localization of *Pentastomum denticulatum*, an arachnoid, which often has man as an intermediate host. The larvae of this parasite may reach the lung via the blood or the lymphatic system and die there (or in the liver), whereas ova of *Taenia solium* are more frequently found in the brains and in the muscles.—*Zur Differentialdiagnose der parasitären Lungenerkrankungen. Cysticercose-Pentastomiasis, F. Weiser, Beitr. z. Klin. d. Tuberk., 1942, 98: 239.*—(*G. Simmons*)

Ornithosis.—Ornithosis is a virus disease. Infections in human beings may occur by direct contact with infected birds, by inhalation of infected droppings or by contact with an infected person. The incubation period is probably eight to fourteen days. The onset of the disease is usually abrupt, with high fever, headache, malaise, nonproductive cough. The physical findings are fluctuating fever with relative bradycardia; rose spots have been observed. The pulmonary findings may vary from normal to frank consolidation, often migrating from one lobe to another. The white blood cell count is elevated at the onset but later on leucopenia may develop. The pulmonary roentgenological findings may vary from increased hilar markings to migratory, patchy infiltrations or pneumonic consolidation. The duration of the febrile course varies from one to five weeks. The very severe form is accompanied by cyanosis and toxic cerebral symptoms. It ends fatally in 35 to 45 per cent. The most important diagnostic procedure is the isolation of the virus from the sputum by inoculation into mice. Until the fourth to tenth day the virus can be isolated from the blood. If no sputum is available the complement fixation test is the most reliable diagnostic procedure provided no infection with the virus of lymphogranuloma venereum is present since these viruses are antigenically related. There is no specific treatment for ornithosis. Experimental evidence points to penicillin as a valuable aid in its therapy while sulfonamides are ineffective. During the past two years 6 sporadic cases of atypical pneumonia were observed in Philadelphia in which a diagnosis of ornithosis could be made by means of the complement fixation test. A history of direct contact was obtained in 2 cases, in the remaining 4 cases daily exposure to pigeons had occurred. The sera of 14 pigeons captured in Philadelphia were tested for the complement fixation titre to ornithosis. The test was positive in diagnostic dilutions in 6 pigeons. Infection of pigeons with ornithosis averaging 40 to 50 per cent is probably of universal occurrence. While cold agglutinins are frequently found in atypical pneumonia they could not be demonstrated in cases of ornithosis.—*Ornithosis as a Cause of Sporadic Atypical Pneumonia, D. C. Levinson, J. Gibbs & J. T. Beardwood, Jr., J. A. M. A., December 23, 1944, 126: 1079.*—(*H. Abeles*)

Treatment of Ornithosis.—A 43 year old man who had a loft of homing pigeons took ill with fever, generalized aching and abdominal discomfort. Physical examination revealed slight impairment of mental coördination, photophobia, atypical consolidation of the left lower lobe, splenomegaly, gaseous abdominal distention and relative bradycardia. The white blood cell count was 9,200. X-ray examination of the chest revealed a central, pneumonic infiltration in the left lower lung. A specimen of the patient's serum obtained on the fifth day of his illness fixed psittacosis antigen in a dilution of 1:256. On the fifth day of his illness the patient was started on 100,000 units of penicillin daily. After the fifth day of treatment the temperature was normal. One month after the onset of his illness the patient returned to work. The virus of ornithosis was recovered from the spleen of one of the pigeons of the patient's loft.—*Human Ornithosis Treated with Penicillin, F. E. Turgasen, J. A. M. A., December 30, 1944, 126: 1150.*—(*H. Abeles*)

Lymphogranuloma Venereum Virus Infection of Respiratory Tract.—After reviewing briefly the literature on experimental and human respiratory infection with this virus, the author refers to his experiences with the entity. In acute cases, whether by buccal or genital invasion of the virus, he has found in some cases the coexistence of tracheobronchial signs, and in chronic cases, such as vulvar erosion, rectal stenosis, peno-scrotal elephantiasis, signs of intense pulmonary fibrosis and involvement of the mediastinal lymph nodes, etc., without its being possible to relate it to tuberculosis. A case history is given of a 46 year old mechanic who for some years had had an ulceration of the gum, slowly growing and resistant to all treatment. A positive Frei test was obtained and biopsy revealed lymphogranuloma virus in large quantities. The patient died of a hemorrhage from rupture of a branch of the internal maxillary artery, caused by extensive ulceration. Necropsy showed intense fibrosis and nodulation of the right lung and marked fibrosis in the alveolar septa, formation of fibrous cortical and subcortical nodules and fibrosis and thrombosis of the vascular system in the left lung. The gum ulcer and the cervical and mediastinal lymph nodes showed intense fibrosis. Great quantities of virus were found in all tissues studied. No evidence of cancer or tuberculosis was found. The author believes pulmonary infection by lymphogranuloma virus can be by direct mucosal extension from a lesion high in the respiratory tract, by the lymphatics or by the circulatory system in which latter case the early symptoms are of the catarrhal type.—*Infeccion del aparato respiratorio por el virus linfogranuloma venereo (L. V.), W. E. Coutts, Rev. chilena de hig. y med. prev., January, 1944, 6: 163.*—(*J. S. Peterson*)

Acute Interstitial Fibrosis of Lungs.—This report adds another case of this rare pulmonary disease to the 4 already reported by Hamman and Rich. A 47 year old Italian-born male was admitted to the Peter Bent Brigham Hospital suffering from severe dyspnea and a dry cough. Physical examination revealed only a few transient râles in the left axilla, but the heart was found to be slightly enlarged to the left and the pulmonic second sound was louder than the aortic. The fingertips were clubbed. The patient had been in good health until four months before admission, when the dyspnea was first noted. X-ray films of the chest showed extensive fine mottling of both lungs most marked in the upper lobes, findings suggestive of miliary tuberculosis. The tuberculin test (first strength), however, was negative and no tubercle bacilli were found in the sputum. A few days after admission fever developed and the respiration became rapid with marked cyanosis. Both legs became mottled and cool below the mid-calf. Many more petechia appeared over the abdomen and anterior chest. Although oxygen administration afforded temporary relief from the air hunger the patient died in respiratory failure eight days after admission. Two days before death a pleural friction rub was heard in the right axilla. At autopsy both pleural cavities were found to be obliterated by moderately dense fibrous adhesions. On sectioning, the lung surfaces presented the mosaic appearance of diffuse fibrosis with interspersed areas of emphysema and pneumonic consolidation. Microscopically there was striking and extensive interstitial fibrosis throughout both lungs. The alveolar walls were found to be greatly thickened by recently formed connective tissue and capillaries. The aveoli were lined by very prominent epithelium and some contained cell debris and phagocytes. The bronchioles were relatively free from exudate. There was no bronchiectasis. Many of the small pulmonary vessels showed obliterative fibrosis. There was no evidence of tuberculosis. Except for some hypertrophy of the wall of the right ventricle the heart was essentially normal. The aorta contained extensive friable antemortem thrombus overlying and entering several of the intercostal arteries. The right iliac artery was occluded by adherent thrombus; a free thrombus partly

occluded the left iliac artery. Infarcts were found in the kidneys and pancreas. The resemblance of the present case to those described by Hamman and Rich is striking. The proliferation and prominence of the lining alveolar epithelial cells seem identical with the changes mentioned by the above authors. Apart from focal areas of terminal bronchopneumonia there were no stainable bacteria in the pulmonary tissue and there was no residuum of the type of inflammation usually produced by pyogenic bacteria. These negative findings also characterized the 4 previously reported cases. The etiology of the underlying pulmonary disease remains obscure. (Illustrated.)—*Report of a Case of Acute Interstitial Fibrosis of the Lungs, H. Eder, C. Van Zandt Hawn & G. Thorn, Bull. Johns Hopkins Hosp., April, 1945, 76: 163.*—(*J. S. Woolley*)

Coccidioidin Tests and Pulmonary Findings.—Only since 1937 has it been recognized that *C. immitis* can cause a wide-spread, benign, pulmonary infection as well as the more severe coccidioidal granuloma with its 50 per cent mortality. The demonstration that the minor respiratory illnesses often associated with erythema nodosum and commonly called "Valley Fever," from its prevalence in the San Joaquin Valley of California, were due to *C. immitis* has stimulated wide-spread epidemiological study. A variety of pulmonary manifestations have been reported in early coccidioidomycosis, namely, bronchial pneumonia, subacute and chronic cavitation resembling tuberculosis and calcified pulmonary lesions. The relationship of healed and calcified pulmonary lesions to positive coccidioidin skin tests forms the basis of this report. Skin testing for sensitivity to coccidioidin as a diagnostic aid or in epidemiological surveys has demonstrated a surprisingly high incidence of reactors depending largely on the locality of the study. In fact, the testing of the school children of the Pima Indian Reservation of Arizona yielded such a high incidence of positive cutaneous reactions (90 per cent) that the test material was first thought to be nonspecific. However, the same antigen, when used in testing Philadelphia children, gave no positive reactions. The clinical material for the present study came from Arizona, New Mexico and the southern part of California. For routine skin tests, 0.1 ml. of a 1:100 dilution of coccidioidin (Smith) was used and the tests were read, together with controls, at twenty-four and forty-eight hours. An area of erythema of 1 cm. or more was accepted as a positive reaction. Purified protein derivative was used for the tuberculin skin tests. The lungs of autopsied cases were removed intact for roentgenological studies. In this way calcified and fibrotic areas were localized and then excised from the lungs and peribronchial lymph nodes. One-half of the excised material was treated and injected into the testicles of guinea pigs and the remainder was decalcified and sectioned. Results: skin tests with coccidioidin were performed on 1,165 patients. Of these, 302, or 25.9 per cent, were positive reactors. According to geographical distribution the incidence of infection was, in part, as follows: The San Joaquin Valley, 62.8 per cent; Arizona, 28 per cent; and Texas, 35.7 per cent. These figures agree rather well with the known distribution of endemic areas of coccidioidomycosis. Approximately 400 of these cases were tested with tuberculin. Comparative studies showed no cross-antigenic relationship between tuberculin and coccidioidin. Skin antigens of other fungi (torula, aspergillus, blastomyces and sporotrichum) prepared in a manner similar to that for coccidioidin produced no skin reactions in patients sensitive to coccidioidin. Thirty-six patients upon whom skin tests had been done were examined by autopsy. Twenty-five had not reacted to coccidioidin. Four of these showed no calcified lesions. The remainder exhibited calcified areas of different sizes in various parts of the lung mostly in moderate numbers. Most of these lesions were interpreted as residual evidence of healed primary tuberculous foci. The lungs of 3, however, suggested a healed miliary type of tuberculosis.

Tubercle bacilli were not found in material from these cases. Two cases dying of advanced coccidioidal granuloma were also negative to coccidioidin. This is not an infrequent finding. Eleven cases with a positive coccidioidin skin reaction dying of intercurrent disease were seen at autopsy. All showed some areas of calcification (or encapsulated necrosis). In 9, endospores or spherules were found but only one positive culture for *C. immitis* was obtained. None showed tubercle bacilli. No clear clinical histories suggestive of coccidioidomycosis infections had been obtained in these positive reactors. The calcified lesions were indistinguishable grossly from similar lesions of tuberculosis except that apical scars were not a part of the healed or arrested pulmonary phases of coccidioides. The histological pattern was essentially that of tuberculosis except for the presence of the spherules. The centres were caseous and contained small calcified particles and faint outlines of necrotic lung tissue. Each lesion was surrounded by a dense, hyalinized, fibrotic capsule, external to which were collections of lymphocytes. The spherules, some containing endospores, were located in the capsules. In none of the animals injected with material from the healed lesions was a lesion of coccidioides or tuberculosis demonstrated. Undoubtedly most of the healed lesions were of tuberculous origin. This would indicate that in healed tuberculosis, as well as healed coccidioidomycosis, the organisms are dead. This is another point in which these two diseases resemble one another. (With 5 plates.)—*Healed or Arrested Pulmonary Coccidioidomycosis: Correlation of Coccidioidin Skin Tests with Autopsy Findings, E. M. Butt & A. M. Hoffman, Am. J. Path., May, 1945, 21: 485.—(J. S. Woolley)*

Pulmonary and Meningeal Torulosis.—Very few cases of infestation of the lung by yeast cells are published. The author presents one case. The first symptoms experienced by the patient, a woman of 45 years, were severe headache, nausea and projectile vomiting. She had marked papillary edema, moderate dyspnea and cough. On auscultation subcrepitant and sibilant râles were heard over the middle portion of the right lung. The cerebrospinal fluid revealed yeast-like fungi which were diagnosed as *Torula histolytica* (*Cryptococcus histolyticus*). In the culture the fungus grew in round cells with double membranes by budding only. The patient died in delirium. The autopsy showed abundant exudate in the subarachnoid spaces which was formed by peculiar polynuclear cells and macrophages. In the right lung there was an abscess containing thick pus. In its wall and also in the neighboring pulmonary tissue isolated round fungus cells with double membranes were found. The tissue reaction to these fungus cells was similar to that observed in the infectious granulomata. Primary pulmonary torulosis is rare, whereas the secondary form associated with cerebral infections is more frequent. This, the patient presented. Pulmonary torulosis may simulate an abscess of the lungs. The presence of yeast cells in the exudate and the absence of the diagnostic elements of tuberculosis or neoplasm can lead to the diagnosis. The value of the specific vaccine in the treatment has yet to be proved.—*Estudio clinico y anatomo-patologico de un caso de torulosis meningoencefalica y pulmonar, T. Castellano, Rev. Asoc. méd. argent., November 30, 1944, 58: 1051.*—(*W. Swienty*)

Nontuberculous Pulmonary Calcification.—In the central eastern half of the United States there is an area of high prevalence of calcified pulmonary lesions. Many of these cases are negative to tuberculin and to coccidioidin. Tuberculin, histoplasmin tests and chest roentgenograms were done in 3,105 student nurses in Minneapolis and St. Paul, Minnesota, Kansas City, Missouri, Kansas City, Kansas, Detroit, Michigan, and Philadelphia, Pennsylvania. The tuberculin tests were performed with Purified Protein Derivative, the histoplasmin tests with 0.1 cc. of a 1:1,000 dilution of a filtrate of broth culture of *Histo-*

plasma capsulatum. Seven hundred and eleven (22.9 per cent) showed a positive reaction, 61 (2.0 per cent) a doubtful reaction to histoplasmin. Two hundred and ninety-four had pulmonary calcifications. Of these, 21.4 per cent reacted to tuberculin. Of the remaining 231, 206 (70.1 per cent) had a positive or doubtful histoplasmin reaction. Only 25 (8.5 per cent) had a negative reaction to both tuberculin and histoplasmin. Among the nurses who reacted only to tuberculin, 10.4 per cent showed pulmonary calcifications; among those who reacted only to histoplasmin, 31.1 per cent showed calcifications; among those who reacted to both antigens, 36.1 per cent showed calcifications; among the 2,141 nurses who were negative to both antigens, only 25 (1.2 per cent) had pulmonary calcifications. The author draws the following conclusions: (*a*) Mild, probably subclinical infection with *Histoplasma capsulatum* (or an immunologically related organism) is widely prevalent in certain states and relatively infrequent in others. (*b*) In general, those states in which the frequency of reactions to histoplasmin is high are those in which pulmonary calcification is also high. (*c*) A very high proportion of the pulmonary calcifications observed in roentgenograms of tuberculin-negative persons is due, not to tuberculosis, but probably to histoplasmosis. —*Nontuberculous Pulmonary Calcification and Sensitivity to Histoplasmin, C. E. Palmer, Pub. Health Rep., May 11, 1945, 60: 513.*—(*G. C. Leiner*)

Echinococcus.—A case of echinococcus cyst of the heart is presented. The clinical diagnosis is based on characteristic roentgenoscopic and roentgenographic evidence of a calcified cyst in the heart wall along with electrocardiographic evidence of myocardial changes and a positive intracutaneous test with echinococcus antigen in the absence of other types of cestode infestation. (Authors' Summary)—*Echinococcus Cyst of the Heart, J. Zizmor & M. M. Szucs, Am. J. Roentgenol., January, 1945, 53: 15.*—(*P. Lowy*)